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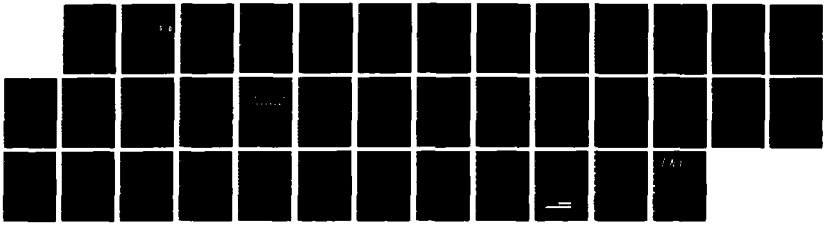

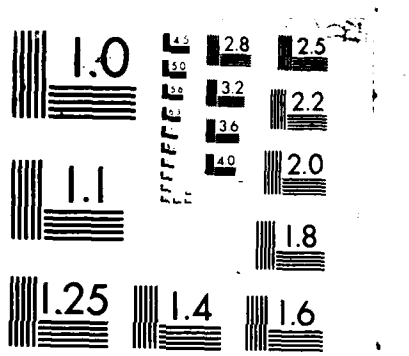

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MUSCLE WATER AND ELECTROLYTE BALANCE DURING CHRONIC EXPOSURE AND WORK IN THE HEAT

ANNUAL/FINAL REPORT

AUTHORS: David L. Costill, Ph.D.
Lawrence E. Armstrong, Ph.D.
Douglas S. King, Ph.D.
William J. Fink, M.S.

DATE: May 31, 1985

SUPPORTED BY
U.S. ARMY MEDICAL RESEARCH AND DEVELOPMENT COMMAND
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HUMAN PERFORMANCE LABORATORY
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In an effort to assess the effect of acute exercise and heat acclimation on muscle metabolism and electrolyte balance, three projects were conducted. The first investigation (Project I) was reported earlier (Annual Report, April 30, 1984) and demonstrated that diets high (399 mEq Na+/day) and low (98 mEq Na+/day) in sodium did not alter the muscle potassium content. The low sodium diet, however, appeared to lessen the subjects' rate of heat acclimation. Project II and III (reported here) examined the effect of heat acclimation on aerobic exercise tolerance in the heat and on subsequent sprint exercise performance. These studies indicate that heat acclimation produces a shift in fuel selection dur'ng submaximal exercise in the heat, with a 47-48% reduction in glycogen use by the vastus lateralis muscle. Leg blood flow studies reveal that heat acclimation does not alter the uptake or release of glucose, free fatty acids or glycerol. Although respiratory exchange ratios and lactate efflux from the muscle revealed a small shift to greater lipid oxidation, these small changes can not explain the sparing of muscle glycogen seen in these studies.

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SUMMARY

This Final Report of our research will outline the final two projects conducted since the submission of our ANNUAL SUMMARY REPORT dated April 30, 1984. Although our first project (PROJECT I) dealt with the influence of diets high and low in sodium on heat acclimation and body potassium balance, Projects II and III focus on the changes in exercise metabolism before and after heat acclimation. Whereas Project II examined the effects of heat acclimation on muscle glycogen use during exercise in the heat, Project III studied the role of leg blood flow and substrate across the leg during exercise.

Project II

The effect of heat acclimation on aerobic exercise tolerance in the heat and on subsequent sprint exercise performance was investigated. Before (UN) and following (ACC) eight days of heat acclimation, ten male subjects performed a heat-exercise test (HET) consisting of six hr of intermittent, submaximal (50% Vo_2 max) exercise in the heat (39.7°C db, 31.0% RH). A 45 s maximal cycle ride was performed before (sprint 1) and after (sprint 2) each HET. Mean (± SE) resting plasma volume increased 9.2% (± 1.7%) following heat acclimation. Muscle glycogen use during the HET was lower following acclimation (ACC = 33.0 ± 5.0; UN = 57.1 ± mmol/kg, $P < 0.05$). No differences were noted between the UN and ACC trials with respect to blood glucose, lactate (LA), or respiratory exchange ratio. During the UN trial, total work output during sprint 2 was reduced compared with sprint 1 (24.01 ± 0.80 vs. 21.56 ± 1.18 kJ, $P < 0.05$). This reduction in sprint performance was associated with an attenuated fall in muscle pH following sprint 2 (6.86 vs 6.67, $P < 0.05$), and a reduced accumulation of LA in the blood. No differences between sprints 1 and 2 were observed for the ACC trial in total work, muscle pH, or blood LA. These data indicate that heat acclimation produced a shift in fuel selection during submaximal exercise in the heat. The observed sparing of muscle glycogen may be associated with the enhanced ability to perform highly intense exercise following prolonged exertion in the heat.

Project III

Theeffects of heat acclimation on leg blood flow, substrate exchange in the leg and muscle glycogen use were studied in eight men. Before and following eight days of heat acclimation the men performed a 60 min heat tolerance test (HTT). A 48% reduction in muscle glycogen use during the HTT was observed as a consequence of the acclimation regimen. Despite this marked reduction in the muscle's reliance on glycogen, there was no significant increase in glucose or fatty acid uptake by the legs, nor was there a significant shift in lipid oxidation to account for the reduced rate of glycogen use.

FOREWORD

For the protection of human subjects the investigators have adhered to policies of applicable Federal Law 45CFR46 and AR70-25.

TABLE OF CONTENTS

Introduction

The process of heat acclimatization has been extensively studied with respect to changes in the body fluid spaces, central circulation and temperature regulation (1,2,3,4). Little is known, however, regarding any adaptive changes which may occur at the tissue (muscle) level.

The importance of muscle glycogen in determining endurance performance has been established (5). Exercise in the heat has also been observed to result in an increased utilization of muscle glycogen compared with exercise in a cold environment (6). The primary objective of Project II was to study the effect of heat acclimation on the utilization of muscle glycogen during prolonged submaximal exercise in the heat. The effect of heat-exercise stress on the ability to perform highly intense exercise was also investigated in subjects before and after heat acclimation. Project III, on the other hand, assessed the contribution of blood borne substrates to muscle metabolism during a standard exercise bout before and after heat acclimation.

PROJECT II

Materials and Methods

Subjects and Design. Ten healthy, untrained males volunteered to serve as subjects in this study after being informed of all risks and stresses associated with these experiments. Written consent was obtained from each subject. A maximal oxygen uptake test was performed before, and two days following the second heat-exercise stress trial. Two of the subjects were engaged in heavy resistance training, while the remaining subjects were not engaged in any regular, vigorous physical acitvity. Subject characteristics are presented in Table 1.

All trials were conducted during the months of January, February, and March to reduce any natural heat acclimatization. Environmental temperatures during these months averaged -6.8, 1.6, and -1.8^{o}C, respectively.

Prior to (UN), and following 8 days of heat acclimation (ACC), the subjects performed a six hour heat-exercise test (HET) and sprint exercise tests as described below (Fig. 1). The men reported to the laboratory after an overnight fast. A pre experiment nude weight was obtained, and a thermister probe (Yellow Springs Instruments) was inserted 10 cm into the rectum. During all heat exposures, the subjects were dressed in athletic shorts and shoes. An infusion set was introduced into an antecubital vein and was kept patent with 0.9% saline.

Sprint Exercise Test. The subjects performed a maximal 45 s ride on a hydraulically braked isokinetic cycle ergometer (Lumex Corporation, Ronkonkoma, N.Y.) prior to heat exposure (sprint 1) and within one min after exiting the environmental chamber following the HET (sprint 2). The pedalling rate was set at 90 rpm for all sprint rides. The ergometer was calibrated before the experiment with known torques. Total work accomplished during the 45 s exercise was measured by digitizing the curves obtained from a chart recorder. The fatigue index as defined by Thorstensson (7) was also calculated.

Needle biopsy samples (8) were obtained from the vastus lateralis muscle before and within 5 s following each sprint bout. Muscle samples were quickly frozen in liquid nitrogen and subsequently analyzed for Na+, K+, Cl-, glycogen, and pH. Blood was obtained in heparinized syringes after five minutes of rest, immediately after sprint exercise, and after 5, 10, and 15 minutes of recovery for the determination of hemoglobin, hematocrit, lactate, and blood gas concentrations.

Heat-Exercise Test. Immediately following the fifteen minute post sprint blood sample, the subjects moved into the environmental chamber and began the six hour HET. The chamber temperature was maintained at 39.7 (±0.1)°C dry bulb and 31.0 (± 0.7%) relative humidity. During this exposure, the subjects exercised on a cycle ergometer (Monark) for the first 30 minutes of each hour at an exercise intensity calculated to require 50% Vo_2 max. Respiratory exchange data were obtained from expired gas collected in Douglas bags during the last 10 minutes of each exercise bout. Heart rates were recorded during the final 30 s of submaximal exercise using a cardiotachometer or by auscultation. Rectal temperature was monitored at 5 min intervals throughout the six hours of heat exposure. During the first, third, and sixth hours, blood samples were obtained during the last minute of exercise and subsequently analyzed for glucose and lactate concentrations. Pre and post HET blood samples were also analyzed for Na+, K+, and Cl- concentrations.

During the final thirty minutes of each hour, the subjects rested in the supine position. The subjects were given 100 ml of tap water (40°C) at thirty minute intervals throughout the HET.

At the conclusion of the HET, the subjects exited the chamber and moved immediately to the bicycle ergometer, where they performed another 45 s sprint ride (sprint 2) as described above.

Heat Acclimation. The subjects performed bicycle exercise in the heat for 90 minutes each day for eight days. The exercise required a mean Vo_2 (± SE) of 2.14 (± 0.07 l/min), or 54.7% of the subject's Vo_2 max. During the heat acclimation bouts, the subjects were allowed water ad libitum.

Analytical Methods. Muscle pH was measured using a homogenate technique as previously described (25). After hydrolysis of the muscle glycogen with 2.0 N HCl, glucosyl units were determined with a fluorometric method (11). Muscle electrolyte determinations were performed after lipid extraction with petroleum ether and twenty four hours of extraction in 2.0 N nitric acid. Sodium and K^+ concentrations in blood and muscle were measured in triplicate by flame photometry. Muscle and blood Cl^- was measured in triplicate by coulometric-amperometric titration (9).

Hemoglobin concentration was determined using the cyanmethemoglobin method. Hematocrit was determined with a microcentrifuge. Plasma volume changes were then calculated according to the procedures of Dill and Costill (10). One ml was kept anaerobic on ice for subsequent blood gas and pH determination. Blood glucose and lactate concentrations were determined enzymatically (11,12). Blood pH, PCO_2, and PO_2 were determined with a BMS 3 MK 2 Blood Micro System and PHM 73 pH Blood Gas Monitor (Radiometer, Copenhagen).

The data were analyzed utilizing analyses of variance for repeated measures designs. Significant mean differences were located with the Newman-Keuls multiple comparison test. The $P<0.05$ level of significance was chosen.

RESULTS

Submaximal Exercise in the Heat. Based on changes in hemoglobin and hematocrit, mean resting plasma volume increased 9.2 ± 1.7% during the eight days of acclimation ($P<0.001$, Table 1). Although mean body weight increased 0.51 kg following acclimation, this difference was not statistically significant. Maximal oxygen uptake was not influenced by the acclimation procedure.

Mean exercise Vo_2 during the HET was not different in the UN (1.92 ± 0.08 l/min) and the ACC (1.88 ± 0.09 l/min) trials. The RER was also unaffected by acclimation state (UN = 0.81 ± 0.01; ACC = 0.80 ± .01). Mean exercise heart rate was significantly reduced following heat acclimation (160 ± 3 beats/min vs. 144 ± 3 beats/min). The increment in rectal temperature during the UN trial (1.45 ± 0.15 C) was significantly greater than in the ACC trial (1.13 ± 0.13 C).

Blood glucose concentration did not differ significantly between the UN and ACC trials at any point (Fig. 2). One subject was unable to complete the final 30 min submaximal exercise bout during the UN trial and exhibited severe hypoglycemia (1.6 mmol/l) at 330 min. This subject was unable to perform the subsequent sprint exercise test but was able to perform the entire HET after the acclimation process.

Blood lactate concentration was significantly higher in the UN trial (3.64 ± 0.38 mmol/l) when compared with the ACC trial (2.87 ± 0.42 mmol/l) at 30 min only (Fig. 2). Values obtained at 30 min and post HET were not significantly different from the resting values in either UN or ACC trials.

Resting muscle glycogen concentrations were not different between the UN and ACC trials (Table 2). Muscle glycogen concentration after the HET was significantly greater in the ACC trial than in the UN trial ($P<0.05$). Acclimation thus resulted in a 42% (33.0 ± 5.0 vs. 57.1 ± 5.7, $P<0.05$) reduction in muscle glycogen utilization.

Sprint Exercise. Total work output (Table 3) was significantly reduced during sprint 2 compared with sprint 1 in the UN trial. No such decrement in work output was observed as a consequence of the HET in the ACC trial. Peak torque, torque at 45 s, and percent decline in torque (fatigue index) were not significantly different between the UN and ACC trials, or between the two sprint exercise bouts.

Muscle glycogen use for sprints 1 and 2 was 28.0 ± 3.9 and 14.8 ± 4.0 mmol/kg, for the UN trial. Corresponding values for the ACC trial were 18.0 ± 7.1 and 19.9 ± 5.6 mmol/kg for sprints 1 and 2, respectively. These means were not significantly different from each other.

Pre sprint muscle pH was not significantly altered by acclimation or by the HET, either in the UN or ACC trials (Table 2). During the UN trial, immediate post exercise muscle pH was significantly lower following sprint 1 (6.67) than sprint 2 (6.86). Values for post exercise muscle pH in the ACC trial were 6.73 and 6.77 for sprints 1 and 2, respectively, and were not significantly different.

No significant differences were noted in the pre sprint blood LA concentrations, either between the UN and ACC trials, or between sprints 1 and 2 (Table 4). In the UN trial, blood LA concentrations after 10 and 15 minutes of recovery were significantly lower following sprint 2 compared with sprint 1. Throughout recovery, blood LA concentrations following sprint 2 were lower for the UN trial compared with the ACC trial ($P<0.05$).

No significant differences were observed between the UN and ACC trials for blood pH prior to, or during recovery from sprint exercise (Table 5). Nadir values, ranging from 7.13 to 7.22, were reached at 5 min post exercise in all sprints.

DISCUSSION

The changes observed in plasma volume, exercise heart rate, and rectal temperature demonstrate that the exercise protocol was successful in promoting acclimation to exercise in the heat.

Muscle Glycogen Use. The major finding of this investigation was that muscle glycogen use during three hours of intermittent exercise was markedly reduced following eight days of heat acclimation. These data suggest an alteration in substrate use following repeated days of exercise in the heat. The mechanism(s) responsible for this decreased reliance on muscle glycogen stores during prolonged exercise in a hot environment can, at present, only be surmized. The reduction in muscle blood flow during exercise in the heat (13) results in a reduced delivery of blood borne substrate to the exercising muscle. An increased perfusion of active skeletal muscle following heat acclimation (2,13) may have resulted in an augmented delivery of both glucose and non-esterified fatty acids, and allowed for a reduced dependence on muscle glycogen for energy production.

The reduction in muscle glycogen use during the ACC trial was not associated with any change in RER or blood LA concentration, suggesting that the rate of muscle glycolysis was unaltered following acclimation. In a recent study, Green et al. (14) observed a reduced muscle glycogen utilization during submaximal exercise in a cool environment following three days of a physical training regimen which increased plasma volume by 21%. As was noted in the present study, Green et al. did not observe any difference in blood glucose concentrations or in the RER. These results indicate that there was no shift in the use of carbohydrate and lipid for energy production. Further, these investigators found that muscle concentrations of LA and glucose-6-phosphate after two hours of exercise were unchanged following the three days of training. These data support the hypothesis that the acclimation process did not result in any change in glycolytic flux during prolonged exercise. Hultman (15) has demonstrated that the rate of muscle glycogen utilization is inversely related to the release of glucose from the liver. Thus, the finding of a reduced glycogen use without an apparent increase in lipid oxidation may reflect an enhanced rate of hepatic glucose release and subsequent use by active skeletal muscle.

Plasma catecholamine levels during exercise in the heat have been demonstrated to be higher than identical exercise in a cool environment (16). Although plasma catecholamines were not determined in the present study, the reduced rectal and skin temperatures following heat acclimation might have resulted in decreased circulating levels of catecholamines during exercise in the heat. Winder et al. (17) observed markedly reduced plasma catecholamine levels during submaximal exercise after only one week of endurance training. Thus, lowered plasma catecholamine levels during exercise in the heat may have played a role in the reduced glycogen utilization by improving splanchnic perfusion, thereby stabilizing hepatic function (18), and also through direct effects on the exercising muscle (19).

Sprint Exercise. Total work output was significantly reduced in sprint 2 in the UN trial, while in the ACC trial the subjects were able to maintain sprint exercise capacity following prolonged exercise in the heat. Previous research suggests that thermal dehydration to a body weight loss of 5% has no effect on the performance of a highly intense (30s) exercise bout (20). Thus, the loss of body fluids does not appear to be responsible for the observed reduction in work output. As noted by previous investigators (21,22), prolonged exercise in the heat did not alter the electrolyte content of muscle (data not shown), and no differences were noted between the UN and ACC trials in the serum concentrations of Na^+, K^+, and Cl^-. In addition, no differences were noted in peak torque between the UN and ACC trials during sprints 1 or 2. These data suggest that the excitability of skeletal muscle was not appreciably different between the UN and ACC trials before the final sprint bout.

The higher muscle pH observed following sprint 2 compared with sprint 1 during the UN trial represents a 75.8 nmol/l (35%) reduction of muscle H^+ accumulation. A diminished accumulation of LA in the blood was also noted following sprint 2 in the UN trial. In addition, venous blood pH tended to be higher following sprint 2 for the UN trial. Reductions in sprint exercise performance and in the ability to accumulate LA in the blood and muscle have been observed when muscle glycogen is reduced (23, 24). Jacobs (23) has suggested that anaerobic performance is reduced when muscle glycogen is lower than 40 mmol/kg, due to a relative lack of substrate for the flux generating step of glycogenolysis. It is interesting to note that muscle glycogen approached this level (46.6 mmol/kg) in the UN trial after exercise in the heat. These data suggest that the reduction in sprint exercise performance following exercise in the heat in the UN trial was due to a reduction in the capacity for anaerobic energy release.

In summary, eight days of heat acclimation resulted in a marked reduction in muscle glycogen utilization during prolonged exercise in the heat. This alteration in substrate use during submaximal exercise was associated with an enhanced ability to perform highly intense exercise following heat-exercise stress.

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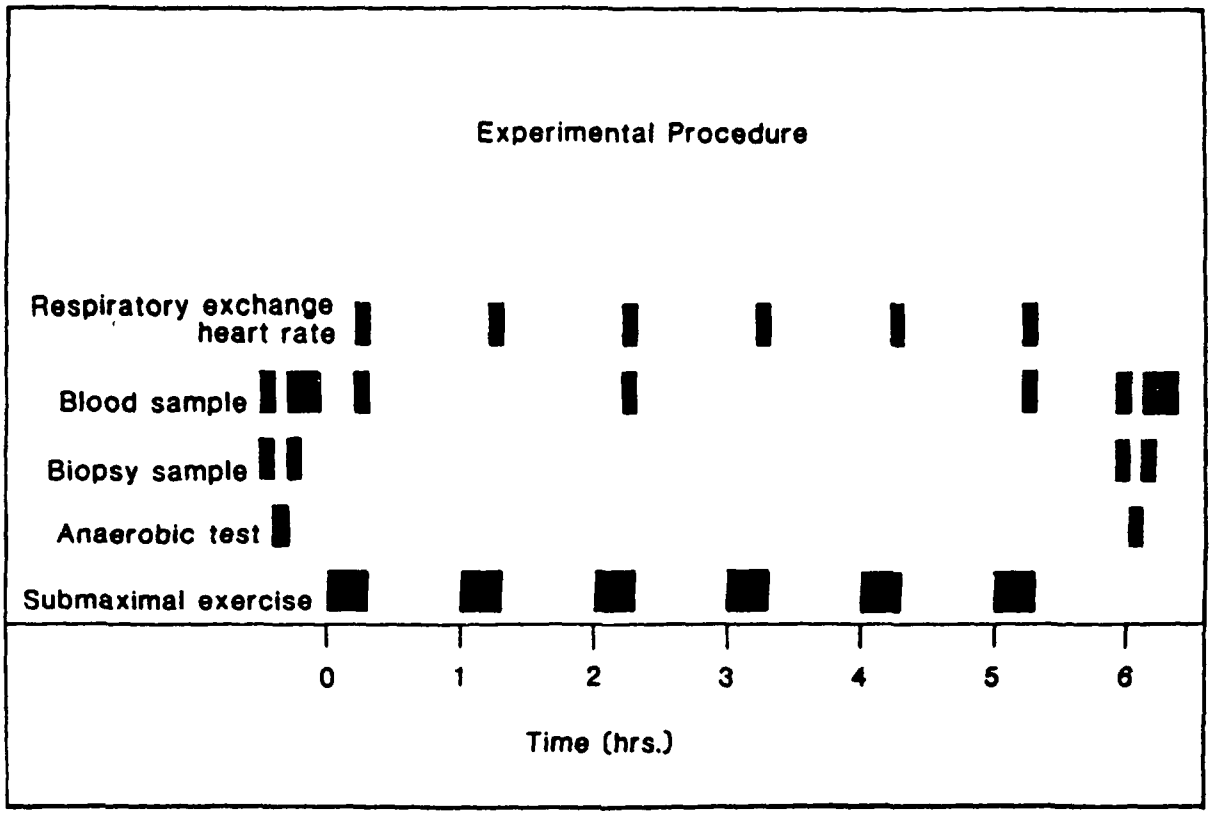
FIGURE LEGENDS

Fig. 1. Experimental procedure.

Fig. 2. Blood glucose and lactate concentrations during six hour HET. * significant difference between UM and ACC trials ($P<0.05$).


Experimental Procedure
Respiratory exchange heart rate
Blood sample
Biopsy sample
Anaerobic test
Submaximal exercise
0
1
2
3
4
5
6
Time (hrs.)

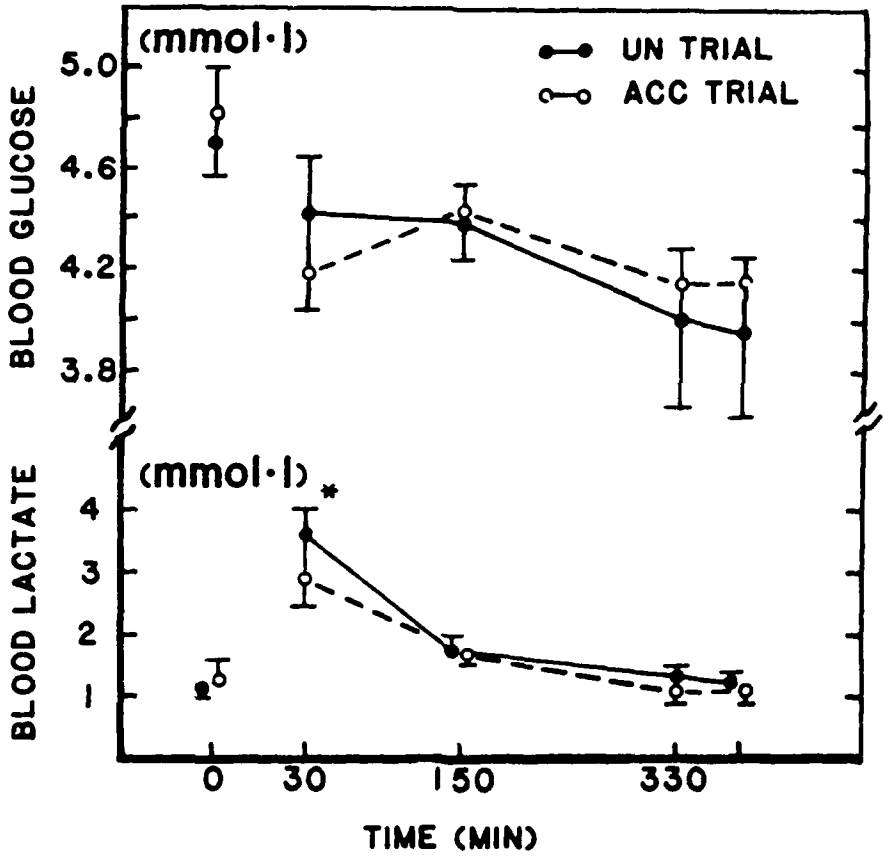

(mmol·l)
UN TRIAL
ACC TRIAL
BLOOD GLUCOSE
5.0
4.6
4.2
3.8
(mmol·l)
BLOOD LACTATE
4
3
2
1
*
0
30
150
330
TIME (MIN)

TABLE 1. Subject characterstics.

Age (yrs)	Ht (cm)	BODY WT (kg)		PLASMA VOLUME (% Change)	$\dot{V}O_2$ max (l/min)	
		UN	ACC		UN	ACC
22.5 ± 1.8	181.1 ± 2.4	79.06 ± 2.36	79.57 ± 2.38	9.2* ± 1.7	3.91 ± 0.12	3.92 ± 0.09

UN and ACC denote unacclimated and acclimated trials, respectively. Values are means ± SE, N=10.

* Significant increase in plasma volume following acclimation ($P<0.05$).

TABLE 2. Muscle glycogen and pH before and after 45 s sprint exercise tests.

		SPRINT 1		SPRINT 2	
SAMPLE		PRE	POST	PRE	POST
GLYCOGEN	UN	131.7 ± 9.1	103.7 ± 9.2	46.6 ± 6.9 *	31.8 ± 5.8 *
	ACC	131.1 ± 9.2	113.1 ± 4.5	80.1 ± 4.4	60.2 ± 4.7
pH	UN	7.15	6.67	7.12	6.86 **
	ACC	7.14	6.73	7.17	6.77

UN and ACC denote unacclimated, and acclimated trials. Sprints 1 and 2 refer to before and after six hour HET, respectively. Glycogen is expressed as mmol/kg wet wt. Values are means ± SE, N=9.

* Significant difference between UN and ACC ($P<0.05$) trials.
** Significantly different from sprint 1, UN trial ($P<0.05$).

TABLE 3. Performance data for 45 s sprint exercise tests.

	SPRINT 1		SPRINT 2	
TIME	UN	ACC	UN	ACC
TOTAL WORK (kJ)	24.01 ± 0.80	24.72 ± 1.02	21.56* ± 1.18	24.67 ± 1.37
PEAK TORQUE (N·m)	82.7 ± 3.3	87.1 ± 3.0	80.6 ± 6.2	84.7 ± 4.8
TORQUE AT 45S (N·m)	35.2 ± 1.4	37.7 ± 2.3	32.2 ± 1.9	36.4 ± 2.5
FATIGUE INDEX (%)	56.9 ± 2.7	56.4 ± 2.8	58.7 ± 3.5	56.2 ± 3.8

UN and ACC denote unacclimated and acclimated trials, respectively. Sprints 1 and 2 refer to before and after six hour HET, respectively. Values are means ± SE, N=9.

* Significantly different from all other means ($P<0.01$).

TABLE 4. Blood lactate concentration before and during recovery from 45 s sprint exercise tests.

	SPRINT 1		SPRINT 2	
TIME	UN	ACC	UN	ACC
PRE	1.12 ± 0.08	1.27 ± 0.13	1.22 ± 0.14	1.12 ± 0.15
0	6.57 ± 1.15	6.01 ± 0.82	5.15 ± 0.81	6.70 * ± 1.23
5	10.33 ± 1.24	11.16 ± 1.02	9.80 ± 1.07	12.40 * ± 1.18
10	11.48 * ± 0.95	11.44 * ± 0.91	9.85 ± 1.08	12.22 * ± 1.09
15	10.90 * ± 0.92	10.88 * ± 0.82	8.69 ± 1.11	10.40 * ± 1.09

UN and ACC denote unacclimated, and acclimated trials, respectively. Sprints 1 and 2 refer to before and after six hour HET, respectively. Concentrations are expressed as mmol/l, and represent means ± SE, N=9.

* Significantly different from sprint 2, UN ($P<0.05$).

TABLE 5. Blood pH before and during recovery from 45s sprint exercise tests.

	SPRINT 1		SPRINT 2	
TIME	UN	ACC	UN	ACC
PRE	7.37	7.37	7.41	7.43
0	7.23	7.25	7.34	7.28
5	7.13	7.14	7.22	7.18
10	7.14	7.16	7.22	7.20
15	7.16	7.19	7.25	7.24

UN and ACC denote unacclimated and acclimated trials, respectively. Values are means ± SE, N=9.

Project III

In light of the findings from Project II a question was raised regarding the effects of heat acclimation on muscle glycogen and blood glucose use during exercise in unacclimated and heat acclimated subjects. Specifically, we were interested in the mechanism that was responsible for the reduced use of muscle glycogen during exercise in heat acclimated men. Since measurements of respiratory exchance indicated that there were similar rates of carbohydrate oxidation in the men before and after heat acclimation, it was theorized that the lower muscle glycogen used was accommodated by an increase in blood glucose uptake by the working muscles. Thus, glucose uptake in the leg was studied during a standard exercise bout before and after a heat acclimation.

Methods and Materials

Eight college-aged men were selected as volunteers after undergoing a physical examination and a maximal oxygen consumption test on a cycle ergometer. The characteristics of these subjects are presented in Table 1. All studies with these men were conducted during the months of November thru February, 1984-85. This period was selected to insure that the men were not heat acclimated. Although they were all normally active, none of the subjects were engaged in a regular cycling or running program at the time of the experiments. The men were informed of all risks and stresses associated with this investigation before giving their written consent to participate.

Each subject performed a 60 min heat tolerance test (HTT) before and after eight days of a heat acclimation regimen. The HTT consisted of cycling on an electrically resisted ergometer (W.B. Collins) at a workload that required an energy expenditure equal to 50% of the subject's maximal oxygen uptake ($\dot{V}o_2$ max). During the heat acclimation regimen the men cycled (Monark ergometer) at 50% Vo_2 max for 90 min per day on eight consecutive days. Each of these exercise bouts and the HTT were conducted in an environmental chamber that was maintained at 39.6°C (SE ±0.2) with a relative humidity of 29.2% (SE ±1.5). Air flow across the head and chest was measured at 0.92 m/sec using a hand held anemometer (FloRite).

During the HTT the following physiological parameters were measured: 1. Rectal temperature using a YSI telethermometer system with the thermister inserted 8 cm beyond the anal sphincter; 2. Leg blood flow using the thermal dilution technique described by Jorfeldt (1) with a single, double lumen catheter inserted in a proximal direction (16 cm) into the right femoral vein. The recording system for injectate and

blood temperatures was provided by American Edwards Laboratories; 3. Muscle biopsies from the vastus lateralis were taken before and within five min after the HTT; 4. Blood samples were obtained from an indwelling catheter inserted into a distal forearm vein. Samples were taken for blood pH, gases, substrates and electrolytes before and at fifteen minute intervals throughout the test. Additional samples were obtained from a radial artery puncture at 30 and 60 min of exercise for similar analysis. The venous catheters were kept patent with a saline drip. 5. Heart rates and respiratory exchange were monitored every 15 min throughout the HTT.

During each 90 min acclimation trial, rectal temperature (YSI thermister) and heart rates were recorded at five minute intervals. Nude body weight was recorded (± 50g) prior to and immediately after each trial. The men were allowed to drink water ad libitum during these exercise bouts, and the volume of water consumed was added to the body weight change to determine the total weight loss. Respiratory exchange gases were collected in a Douglas bag at 15, 60 and 90 min of the trials on days 1, 4 and 8. Exercise was terminated if heart rate exceeded 180 beats per minute or if rectal temperature exceeded 39°C.

Leg blood flows were determined by thermal dilution using a single 5 ml bolus injection. The average temperature of the injectate was 12°C, which produced a mean decrease in blood temperature of approximately 0.5 - 1.0°C. These measurements were made at rest and at 15, 30, 45 and 60 min of the HTT. Four or five measurements were made at each time interval. The highest and lowest values recorded were excluded and the remaining values averaged. In several subjects, abnormally high blood flow readings were observed. Consequently, their leg blood flows (BF) were calculated from the following equations (1): BF = 0.029 (watts) + .67.

The muscle biopsy technique of Bergstrom (2) was modified to include suction (3). The samples were divided, weighed and frozen in liquid nitrogen. At a later time the muscle samples were analyzed for glycogen (3), citrate synthase activity (4), and K+ content (2).

Blood pH, Po_2 and Pco_2 were made using a radiometer system. These values were used to calculate bicarbonate concentration, base excess, and oxygen content. Aliquots of the serum were used to determine the glucose, free fatty acid, glycerol, sodium and potassium concentrations as described in Project II of this report. The values for pulmonary gas exchange were used to calculate the caloric expenditure, carbohydrate use and fat oxidation during the exercise trials.

Results and Discussion

Eight days of heat acclimation. Based on the changes in hemoglobin and hematocrit (Table 4) measured at rest before the two HTT, before and after acclimation, plasma volume was estimated to increase 6.1%. As a consequence of the repeated days of heat-exercise stress, the subjects' mean heart rate and the rise in rectal temperature during the daily bouts were reduced (Figure 1). These changes in plasma volume, exercise heart rate and rectal temperature are all indicative of the adaptations which characterize heat acclimation. There appeared to be no physical training effects as a result of the eight days of moderate exercise (50% Vo_2 max). Maximal oxygen uptake, maximal heart rates and muscle citrate synthase were not significantly different in the pre- and post-acclimation tests (Table 2). Thus, the changes noted during the HTT appear to be associated with adjustments to the heat rather than adaptations in cardiovascular or muscular endurance.

Heat tolerance test. Table 3 presents the mean ±S.E. for oxygen uptake, respiratory exchange ratio, calculated carbohydrate oxidation, leg blood flow, and estimated oxygen uptake in the leg during the HTT before (UN) and after (AC) heat acclimation. Although the pulmonary oxygen uptake was unaffected by the eight days of exercise in the heat, the respiratory exchange ratio tended to be lower in the trial. On the average, the RER was significantly lower after heat acclimation, resulting in a significantly lower rate of carbohydrate oxidation. The difference in total carbohydrate use (g/hr) between the UN and AC trials was, however, only 7.3 grams. Leg blood flow during exercise did not change with heat acclimation, averaging 4.75 and 4.82 l/min for the UN and AC trials, respectively. The oxygen uptake by both legs averaged 1.33 l/min. Thus, approximately 64% of the pulmonary oxygen uptake was being used by the legs. These values for pulmonary Vo_2, leg blood flow and oxygen uptake are in keeping with previous studies which measured these parameters during exercise at this workload (avg. 123 watts).

Muscle glycogen use during the 60 min HTT was significantly reduced ($P<.05$) after heat acclimation (Figure 2). On the average, 67 mmol/kg w.w. of glycogen was used during the first HTT (unacclimated), whereas only 35 mmol/kg w.w. was oxidized during the post-acclimated trial. It is interesting to note that this 47% reduction in muscle glycogen use is similar to the 48% decline in glycogen utilization during exercise in Project II of this report. These findings confirm our original observations that the eight days of exercise in the heat reduced the subjects' reliance on muscle glycogen as a carbohydrate source of energy.

No significant differences were found in the exchange of substrates, waste products or minerals across the leg. Consequently, we have chosen to present only the mean (+S.E.) values from the femoral venous blood samples (Table 4). Blood glucose (femoral vein) was significantly lower ($P<.05$) at all times during exercise after heat acclimation. Although blood lactate tended to be lower during the second HTT, the only significant difference ($P<.05$) was found at 15 min of exercise. It should be noted, however, that the mean values for lactate during exercise are relatively low (3 mmol/l), as would be expected for subjects exercising at 50% Vo_2 max.

Blood free fatty acids (FFA) and glycerol rose steadily during the HTT. In turn, these values tended to be higher during the AC trial compared to the means during exercise in the pre-acclimation HTT (Table 4). In keeping with the lactate values, blood (arterial and venous) pH changed very little during the HTT. Although the difference between the mean pH values was small, there was a significant difference ($P<.05$) between the means at 30 min of exercise. While the arterial (radial artery) pH averaged 7.426, femoral venous blood ranged from 7.391 to 7.326 (Table 4).

Serum sodium concentration was similar in both the arterial (144.3 mmol/l) and femoral venous bloods (145.1 mmol/l). There was, however, a gradual but significant rise in serum sodium over the duration of the HTT in both the UN and AC trials. This finding was in accord with the apparent hemoconcentration associated with sweating and the increase in hemoglobin and hematocrit during the hour of exercise (Table 4). Serum potassium showed a similar pattern of change during exercise, rising from a mean of 4.06 mmol/l at rest (avg. UN and AC) to 4.78 mmol/l at 60 min of cycling (Table 4). Although there were no differences between the UN and AC means for venous potassium concentration, the arterial-venous difference demonstrated that there was a continual release of potassium from the legs during exercise. At 30 and 60 min of exercise, for example, the release (one leg) of potassium averaged 1.38 mmol/min and 1.54 mmol/min, respectively.

As in our previous studies, no changes were found in muscle potassium content either during the HTT or as a consequence of the heat acclimation regimen. Resting muscle potassium averaged 51.8 and 51.7 mmol/100 g of fat free solids in the UN and AC trials, whereas after the 60 min HTT the means were 50.6 and 49.7 mmol/100 g fat free solids, respectively. Thus, despite the 1.46 mmol/min efflux of potassium from the leg during exercise, there was little influence on the muscle potassium content. If we assume that all the potassium released from the leg came from the muscles and that there was 10 kg of muscle involved in this release, then we would have expected muscle potassium to decrease by approximately 8.8 mmol/kg wet wt. or .22 mmol/100 g fat free solid wt. It seems, therefore, that the method for determining muscle potassium is not sensitive enough to identify these small changes in potassium content.

In summary, the most marked findings of this research were the reduced rate of muscle glycogen use during exercise with only a small change in total carbohydrate oxidation and no detectable change in the leg glucose uptake. We also noted a continued efflux of potassium from the leg throughout the 60 min of exercise, but the total loss over that period did not significantly alter the muscle potassium content.

References - Project III

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TABLE 1

CHARACTERISTICS OF THE SUBJECTS (N = 8)

	Age (yr)	Ht (cm)	Wt (kg)	% Fat	Vo_2 max (ml/kg·min)
Mean	24.8	179.6	76.4	13.0	54.5
(±SE)	(1.0)	(1.3)	(1.5)	(1.6)	(2.2)

TABLE 2

Mean (±SE) aerobic capacity (Vo_2 max), maximal heart rate (HR), maximal workload attained during the test for aerobic capacity, and the citrate synthase (CS) activity in the vastus lateralis muscle of the subjects before and after heat acclimation.

	Vo_2 max (L/min)	HR (beats/min)	Workload (Watts)	CS (μmol/g·min)
Before	4.12 (±.11)	190 (±2)	283 (±11)	22.3 (±.9)
After	4.06 (±.13)	190 (±2)	280 (±10)	21.8 (±1.3)

TABLE 3

Mean (+SE) values for pulmonary oxygen uptake (Vo_2), respiratory exchange ratio (RER), calculated carbohydrate oxidation (CHO), leg blood flow, and leg oxygen uptake during exercise in the heat before (UN) and after (AC) heat acclimation.

		EXERCISE TIME				
		15 min	30 min	45 min	60 min	Avg.
Pulmonary Vo_2 (L/min)	UN	1.76 (±.15)	2.10 (±.09)	2.12 (±.11)	2.22 (±.10)	2.05 (±.03)
	AC	1.90 (±.14)	2.18 (±.14)	2.19 (±.11)	2.12 (±.10)	2.10 (±.02)
RER Vco_2/Vo_2)	UN	.85 (±.02)	.86 (±.01)	.84 (±.01)	.83 (±.01)	.844 (±.006)
	AC	.84 (±.01)	.83* (±.01)	.83 (±.01)	.82 (±.01)	.827* (±.005)
CHO (g/min)	UN	1.076 (±.011)	1.372 (±.010)	1.207 (±.009)	1.170 (±.008)	1.201 (±.006)
	AC	1.081 (±.009)	1.150* (±.008)	1.154* (±.008)	1.030* (±.007)	1.080* (±.004)
Leg Blood Flow (L/min)	UN	4.160 (±.330)	4.677 (±.154)	4.895 (±.295)	4.826 (±.192)	4.639 (±.131)
	AC	4.404* (±.290)	4.835* (±.227)	5.034 (±.315)	4.801 (±.196)	4.800 (±.131)
Leg Vo_2 (ml/min)	UN	----	643 (±53)	----	684 (±53)	664 (±35)
	AC	----	665 (±54)	----	676 (±37)	670 (±34)

* Denotes P<.05

TABLE 4

Femoral venous glucose (mean ±SE), lactate, free fatty acids (FFA), glycerol, potassium (K^+), sodium (Na^+), pH, hematocrit (Hct), and hemoglobin at rest and during exercise in men before (UN) and after heat acclimation (AC).

		REST	15 min	30 min	45 min	60 min
GLUCOSE (mmol/L)	UN	5.18 (±.18)	5.30 (±.18)	5.22 (±.22)	5.26 (±.16)	5.19 (±.18)
	AC	4.89 (±.12	4.86* (±.16)	4.79* (±.16)	4.86* (±.16)	4.72* (±.16)
FFA (mmol/L)	UN	.55 (±.09)	.35 (±.05)	.37 (±.05)	.40 (±.05)	.50 (±.05)
	AC	.70 (±.15)	.41 (±.07)	.43 (±.06)	.48 (±.05)	.54 (±.05)
GLYCEROL (mmol/L)	UN	.085 (±.011)	.135 (±.012)	.197 (±.026)	.230 (±.020)	.275 (±.026)
	AC	.092 (±.004)	.161 (±.022)	.226 (±.020)	.258 (±.020)	.298 (±.024)
LACTATE (mmol/L)	UN	.96 (±.08)	2.21 (±.42)	2.47 (±.42)	2.30 (±.40)	1.99 (±.30)
	AC	.91 (±.08)	1.47* (±.27)	2.02 (±.40)	1.82 (±.34)	1.73 (±.24)
pH	UN	7.391	----	7.326	----	7.351
	AC	7.384		7.338		7.355
K^+ (mmol/L	UN	4.08 (±.04)	4.56 (±.07)	4.77 (±.10)	4.74 (±.05)	4.85 (±.05)
	AC	4.03 (±.13)	4.51 (±.16)	4.71 (±.15)	4.59 (±.13)	4.70 (±.12)
Na^+ (mmol/L)	UN	141.6 (±.9)	144.3 (±.5)	144.8 (±.9)	145.0 (±.9)	145.4 (±.9)
	AC	142.7 (±.9)	144.6 (±1.0)	145.5 (±.9)	145.9 (±.9)	146.6 (±.9)
Hct (%)	UN	42.9* (±.5)	----	43.9 (±.7)	----	43.3 (±.6)
	AC	41.3 (±.6)	----	42.5 (±.8)	----	42.6 (±.8)
Hb (g%)	UN	15.7 (±.2)	----	16.1 (±.2)	----	15.9 (±.2)
	AC	15.2 (±.2)	----	15.5 (±.2)	----	15.7 (±.2

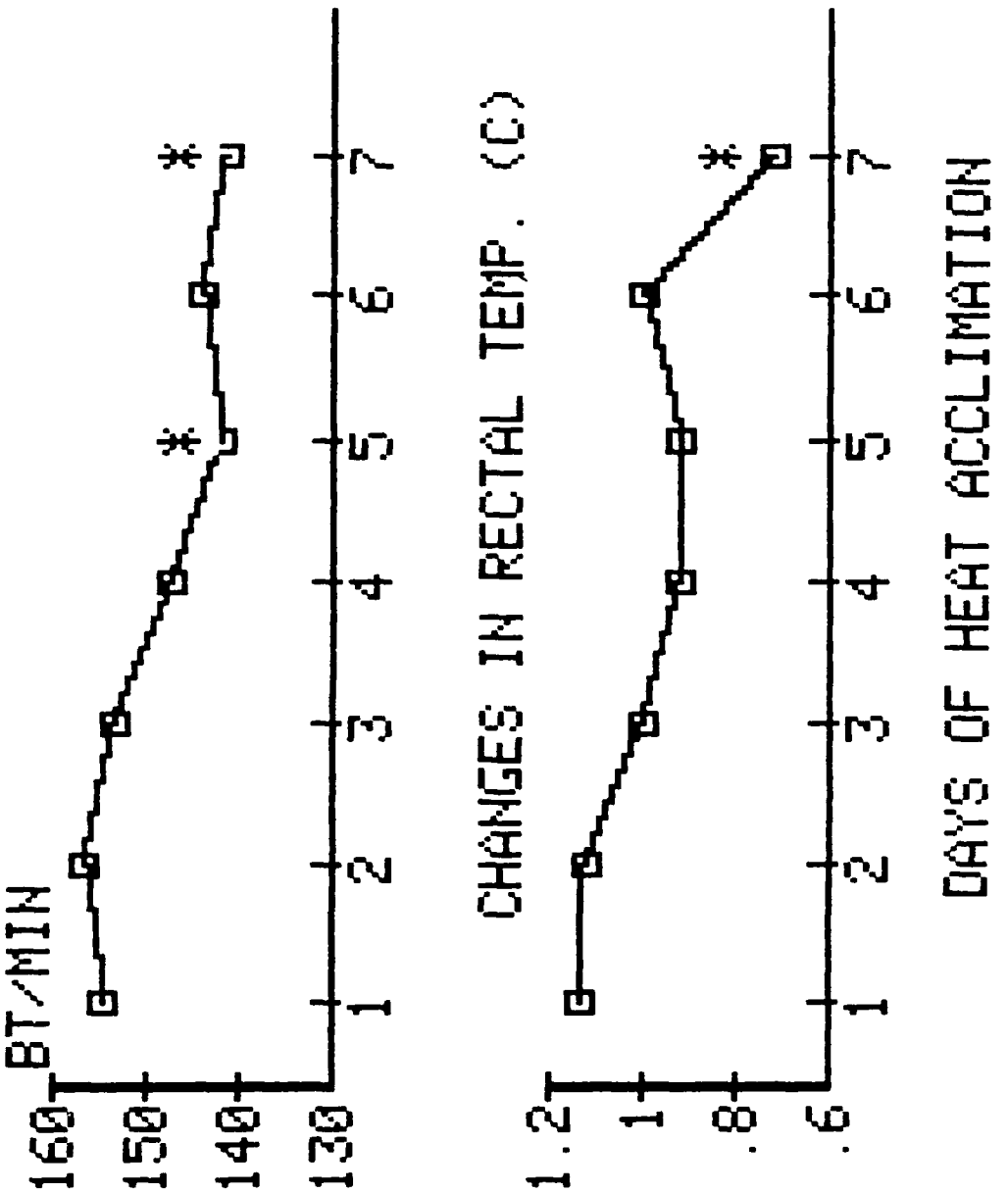
*Denotes a significant difference between means of UN and AC.

Project III
LEGEND OF FIGURES

Figure 1. Peak heart rate (beats/min) and the change in rectal temperature (Pre-Post Exercise) during exercise on each of the days of heat acclimation.

Figure 2. Muscle glycogen content (A) and the amount used (B). Left panel shows the initial (0') and post-exercise (60') values for the pre-acclimation (solid bars) and post-acclimation (stripped columns) trials.

FIGURE 1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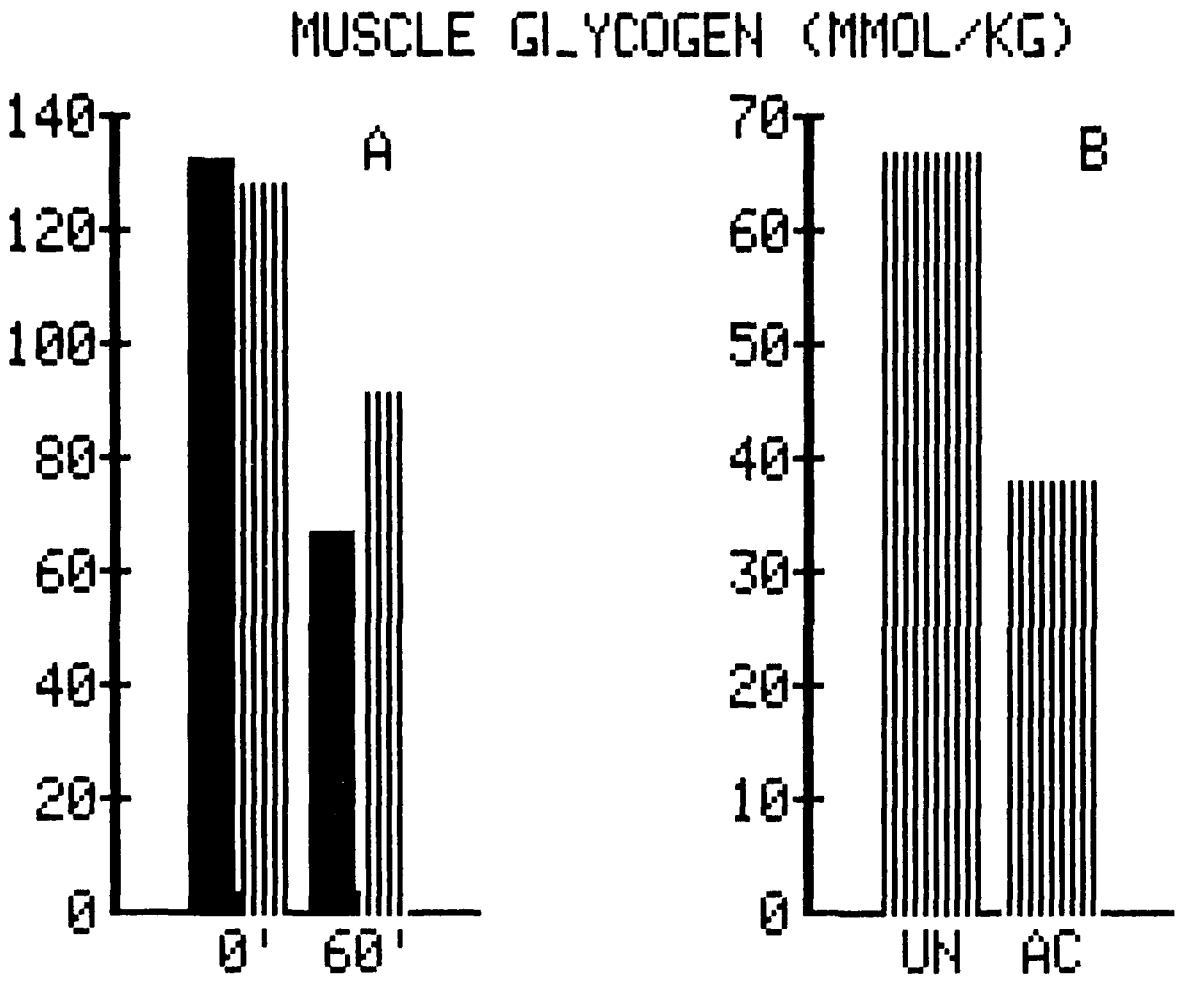
DAYS OF HEAT ACCLIMATION

FIGURE 2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